# Southwestern

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1. Stephens, V. C., *et al.*: J. Am. Pharm. A. (Scient. Ed.), *48*:620, 1959.
2. Salitsky, S., *et al.*: Antibiotics Annual, p. 893, 1959-1960.
3. Reichelderfer, T. E., *et al.*: Antibiotics Annual, p. 899, 1959-1960.
4. Kuder, H. V.: Clin. Pharmacol. & Therap., in press.


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**Plan To Attend . . .**

**SOUTHWESTERN MEDICAL ASSOCIATION**

**42nd Annual Meeting, El Paso, Oct. 20-22**

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## Coming Meetings

Southwestern Medical Association, 42nd Annual Meeting, Hotel Hilton, El Paso, Oct. 20-22, 1960.

American College of Gastroenterology, 25th Annual Convention, Bellevue-Stratford Hotel, Philadelphia, Oct. 24-26, 1960.

Western Association of Railway Surgeons, Annual Meeting, Houston, Oct. 27-29, 1960.

American Fracture Association, Annual Meeting, Mexico City, Oct. 30-Nov. 4, 1960.

Southwest Obstetrical and Gynecological Society, Annual Meeting, Riviera Hotel, Las Vegas, Nev., Nov. 7-8, 1960.

Medical Society of the United States and Mexico, Annual Meeting, Guadalajara, Nov. 8-10, and Mazatlan, Nov. 11-12, 1960.

American Cancer Society, Arizona Division, Ninth Annual Cancer Seminar, Tidelands Motor Inn, Tucson, Jan. 12-14, 1961.

Texas Orthopaedic Association, Galveston, Texas, April 24, 1961.

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SOUTHWESTERN MEDICINE

VOL. XLI, NO. 10 OCTOBER, 1960

# Southwest OB-GYN Society To Meet November 6-8 in Las Vegas, Nev.

The tenth annual meeting of the Southwest Obstetrical and Gynecological Society will be held in Las Vegas, Nevada, November 6, 7 and 8, 1960, with headquarters in the Riviera Hotel.

Dr. Charles J. Newcomb, Tucson, President of the Society, has announced that Dr. Ralph A. Reis, Professor of Obstetrics and Gynecology of Northwestern University Medical School and Editor of Obstetrics and Gynecology, who has been a witty participant in the annual meeting for the last four years, would again serve on the scientific panel.

Other guest speakers will be:

Dr. R. Gordon Douglas, Professor of Obstetrics and Gynecology at Cornell University School of Medicine and Director of the American Board of Obstetrics and Gynecology.

Dr. J. P. Greenhill, Professor of Gynecology at Cook County Graduate School of Medicine and Editor of The Year Book of Obstetrics and Gynecology.

### Other Speakers

Dr. Earl M. Marsh, Assistant Clinical Professor in Gynecology and Obstetrics at the University of California Medical School at San Francisco and Vice President of the American Association of Marriage Counselors.

Dr. John L. Parks, Professor of Obstetrics and Gynecology at the George Washington University School of Medicine and Director of the American Board of Obstetrics and Gynecology.

Other officers of the society are:

Dr. Robert P. O'Donnell, Las Vegas, vice president; Dr. John F. Wanless, San Diego, president-elect; Dr. Raymond J. Jennett, Phoenix, treasurer; and Dr. Zeph B. Campbell, Phoenix, secretary.

Members of the program committee are Dr. Wanless and Dr. Charles T. Franklin, La Mesa, California.

### SCIENTIFIC PROGRAM

**Monday, November 7**

Presiding: John Wanless, M.D., San Diego, President-Elect

9:50 a.m. The Management of the Incompetent Cervix.
R. Gordon Douglas, M.D.

10:30 a.m. Surgical Treatment of Female Sterility.
J. P. Greenhill, M.D.

11:20 a.m. Operative Obstetrics.
John L. Parks, M.D.

12:00 noon An Approach to Psycho-Physiological Gynecology.
Earl M. Marsh, M.D.

1:15 p.m. Round Table Luncheon — Clover Room.
Presiding: Ralph A. Reis, M.D., and Robert C. O'Donnell, M.D.

2:30 p.m. Annual Business Meeting.

**Tuesday, November 8**

Presiding: Paul O. Wiig, M.D., Reno

9:40 a.m. Perinatal Problems.
J. P. Greenhill, M.D.

10:20 a.m. A Conservative Approach to Gynecological Surgery.
R. Gordon Douglas, M.D.

11:10 a.m. Is Prenatal Education of Obstetrical Value?
Earl M. Marsh, M.D.

11:50 a.m. The Significance of Climacteric Bleeding.
John L. Parks, M.D.

1:00 p.m. Round Table Luncheon — Clover Room.
Presiding: Ralph A. Reis, M.D., and D. Dalton Deeds, M.D.

# Santa Fe Seminar

St. Vincent Hospital, Santa Fe
April 26, 1960

**Chairman: Harry D. Ellis, M. D.**
**Case Presentations: Fred Soldow, M.D.**
**Seminar Summary: Bergere A. Kenney, M.D.**

## Anginal Pain

**Dr. Ellis:**

The first part of the program this evening is an attempt to emphasize the importance of distinguishing chest pain of cardiac origin as opposed to chest pain that is not. The two cases to be presented are not good examples of angina pectoris but they are good examples of anginal and non-anginal pain. I have selected these cases because I think they emphasize the difficulty one may have in deciding whether or not a patient's chest pain is serious in import or whether it is only serious to the extent of the discomfort experienced. Dr. Fred Soldow will present our cases in summary form. Dr. Soldow.

**Presentation of Case Summaries: Dr. Fred Soldow:**
**Case No. 1 F. J., Age 52.**

This 52-year-old plumber was seen by his physician February 22, 1957, complaining of epigastric pain. This pain began the previous night and followed a day of heavy exertion. The pain continued for several hours during the night, disturbing his sleep. By morning there was still slight epigastric discomfort and he was somewhat anxious. The patient described the pain as having been high epigastric, severe and steady. There was some burning in the substernal area. There was some aching of the shoulders but no radiation of pain down the arms.

Physical examination revealed normal findings including a normal blood pressure. The EKG, ESR, and SGO-Transaminase were normal.

The family history was negative except for the fact that a brother had died of coronary thrombosis.

### Past Medical History

The past medical history revealed a similar episode of pain following exertion three months prior to this illness. This disappeared in a few hours and the patient did not seek medical advice at that time.

During the week following February 22nd the patient experienced some pain, particularly after meals and during the night. On February 26th, X-ray studies revealed the presence of a diaphragmatic hernia.

On March 3rd, he again experienced severe epigastric pain with radiation to the shoulders and down the arms. The pain persisted throughout the night, not being relieved by Demerol. He was hospitalized March 4th, with a clinical impression of strangulated diagphragmatic hernia. An electrocardiogram taken on this admission to the hospital revealed characteristic changes of myocardial infarction.

**Case No. 2 Age 52.**

This 52-year-old white male first sought medical advice at age 45, because of chest pain in the right parasternal area. This was associated with tenderness over the right fourth and fifth costochondral junctions and the lower third of the sternum.

This pain occurred at rest and was neither produced nor aggravated by exertion. The patient was a heavy smoker. Physical examination at that time was normal as was an electrocardiogram. Over the next seven years he was seen eighteen times by his physician, each time complaining of tightness in the chest and pain either over the lower one-third of sternum, the left parascapular area with radiation into the left upper anterior chest, or with pain confined to the left anterior chest above the level of the fourth rib.

On all these occasions the pain occurred at rest, usually in the late afternoon or evening. The patient denied pain with exertion and actually found that moderate exertion seemed to relieve the pain.

There was no radiation into the jaws or arms

and no dyspnea. He smoked an average of two packages of cigarettes daily.

Electrocardiograms were taken every six months during this period and were always normal. A series of five chest X-rays during this time were likewise normal. Routine examinations on these eighteen visits almost always showed a "trigger" area at the site of the pain.

On November 21, 1959, the patient came to his physician's office without an appointment, stating that during the preceding evening he had had a substernal pain which lasted for fifteen minutes, with subsequent complete relief. On the morning of this consultation the pain recurred in the same location, again lasting ten to fifteen minutes.

At the time of this examination, he had no pain but stated that his chest felt tight as it frequently did. Although the patient could not state how, he insisted that this pain was different than any that he had previously had and he appeared more apprehensive than usual.

Physical examination revealed only tenderness of the left second and third costochondral junctions and the lower sternum. An electrocardiogram showed no change in basic pattern but there was slight depression of RST, V-1 through 4, which was a definite change from previous tracings.

Because of the patient's severe apprehension, the suspicion of cardiac disease was not given him. However, he was placed on Peritrate and given a supply of nitroglycerin to be taken sublingually in the event of a recurrence of the pain. He was likewise cautioned to rest over the weekend and to call if he had any pain not relieved in ten minutes by nitroglycerin.

The following morning at 11:00 A.M. he fell to the floor and was dead by the time of the arrival of his physician, approximately eight minutes later.

**Dr. Ellis:**

Thank you, Dr. Soldow. Dr. Richard Landmann is going to discuss these cases and some of the more practical aspects of the problem of diagnosis of anginal pain. Dr. Landmann.

**Discussion: Dr. Richard Landmann:**

Dr. Ellis asked me to speak about the diagnosis of angina pectoris without going into the differential diagnosis which will be discussed later by Dr. Angle.

I would begin by saying that not all chest pain is angina pectoris. As you know the term angina pectoris was originally coined by Heberden in 1768 to denot a very distinctive form of pain accompanied by a sense of strangling and anxiety. Heberden used the word angina which means strangling to distinguish this pain from what he termed dolor pectoris. He also made mention of psychic components accompanying this pain, the most striking of which was a fear of unpending death.

Clinically, we must remember that angina pectoris is not a disease entity but a clinical syndrome consisting of paroxysmal attacks of characteristic pain or discomfort usually located retrosternally and radiating to the precordium and the left upper extremity, especially the inner side of the arm. The pain often travels down to the fingers, sometimes to an area between the shoulder blades, and sometimes up into the left cheek.

**Precipitated By Effort**

It is precipitated by effort either physical or psychic and disappears with rest or is promptly relieved by nitrites and may be followed by sudden death.

Friedberg lists the essential diagnostic features of angina pectoris as 1) the paroxysmal occurrence of pain, 2) its brief duration, 3) its characteristic location and radiation, 4) its precipitation by effort and relief by rest, and 5) its relief by nitrites.

In order to establish the diagnosis a detailed history is a prerequisite. The patient must be asked to describe the pain in as much detail as he possibly can and one will find that the pain description given by the patient usually consists of a constricting pain, a pressing or boring feeling or an expanding pain. Sometimes it is described as a sense of a band tight about the chest or just a feeling of tightness. Occasionally, it will be described as a burning or heartburn and occasionally one will hear it described as a weight over the chest or a sense of choking. These sensations may be mild, severe or excruciating.

It is of interest that usually with typical anginal pain, no dyspnea is noted. Often the pain will compel the patient to remain as quiet as possible, stopping suddenly whatever he may be doing. This is rather interesting in that other types of pain do not necessarily compel the patient to remain quiet but rather provoke him to thrash or move about.

I think that a prerequisite for the diagnosis of angina pectoris is also the duration of the attack. This is rarely above fifteen minutes.

Occasionally, a patient will have typical pain at night while he rests in bed. If such happens one should be able to obtain a history of attacks with effort during the day. Failing this, I do not think one can apply the term angina decubitus.

The precipitating causes of typical angina of effort are usually bodily exertion, exposure to cold, exposure to wind, walking uphill or walking faster than usual.

Many people with angina of effort have found their own speeds and will not exceed it in walking for they know that otherwise they will have an attack.

**Overeating**

Often an attack will be prompted by overeating. One may obtain a history that a business man can walk very well to his favorite restaurant at lunch time but will have an attack walking the same distance back after his meal.

Emotional strains are not uncommon as a cause of angina of effort. Other precipitating causes may be hypoglycemic attacks, the administration of thyroid extracts, excessive smoking, and apparently nightmares.

Now as to the definite diagnosis of angina pectoris the patient's history is one of the most important factors. Physical examination can be entirely negative, however, if evidence of cardiac disease is present, the diagnosis of angina pectoris is supported.

In many cases nothing will be found. The pulse, blood pressure, and respiration will be quite normal. In such instances, we resort to such tests as the exercise tolerance test, the master-two-step test, the hypoxemia test by which the subject breaths a mixture of carbon dioxide, oxygen, and 90% nitrogen for about twenty minutes unless cardiac pain intervenes, or an epinephrine test.

An electrocardiogram is taken before and after these tests and in many cases of true angina typical changes, such as depression of ST segments greater than one mm., especially in leads II and V-5., are produced.

The epinephrine test has been given up by many people because of the danger of severe reactions, including the precipitation of an acute myocardial infarction.

If we are going to apply the foregoing data to the cases presented tonight I would say that case number one certainly did not have angina pectoris.

The pain in case number one began at night, although following a day of heavy exertion. However, the pain continued for several hours which is certainly not typical. We know that he had a diaphragmatic hernia which certainly could explain his pain. However, on March 3rd the pain was much more severe and persisted. This pain was due to myocardial infarction and was not the typical pain of angina pectoris.

Case number two had pain over his costochondral junctions of the fourth and fifth ribs and the lower third of the sternum which occurred at rest and was not influenced by exertion.

This certainly does not reflect the typical pain of angina pectoris.

"Trigger" areas can confuse one and occasionally are due to angina or myocardial infarction. I have previously reported two such cases with electrocardiographic changes incident to underlying cardiac disease.

This patient stated on November 21, 1959 that he had again some substernal pain which lasted for fifteen minutes with complete relief subsequently and then a recurrence the following morning of the pain which again lasted for ten or fifteen minutes. Unfortunately, it is not known whether the patient had exerted himself shortly before these pains occurred, however, the patient did volunteer the information that this pain was different from the ones previously experienced and he appeared much more apprehensive than usual.

I assume that the electrocardiogram was taken without exercise. There was definitely a depression of RST, V-1 through 4 which may very well have been due to coronary insufficiency. It was later proved that this man most likely died from an infarct, and the two episodes of pain immediately before death constitute definite angina pectoris.

**Dr. Ellis:**

Thank you, Dr. Landmann. Are there questions or comments concerning Dr. Landmann's discussion? If not, we will go to Dr. Richard Angle who will discuss the differential diagnosis of anginal pain. Dr. Angle told me the other day that he sees two to three patients a week complaining of pain in the chest that turns out not to be anginal

pain. Certainly this differential diagnosis is very important in clinical practice.

**Differential Diagnosis: Dr. Richard Angle:**

I am sorry to say that the second patient presented this evening was a patient of mine. Nevertheless, I feel that originally his pain was not angina. This is likely a man who had non-coronary pain for a long time and who also had progressive coronary artery disease which became evident only shortly before his death.

In considering the causes of chest pain, it is well to remember that errors of commission may be just as harmful as errors of omission. It is wrong in my opinion to incorrectly place a diagnosis of angina pectoris and hence a diagnosis of serious heart disease on a healthy patient as it is to conconclude that anginal pain is of non-cardiac origin. Many patients have needlessly been restricted and deprived of life insurance and suffered serious mental anguish by the unwarranted diagnosis of angina. It certainly behooves every physician to make the diagnosis with certainty.

There are certain factors that are important in considering the differential diagnosis of chest pain, especially the character of the pain, its manner of onset, and its manner of relief.

Of other importance are the age and the sex. It is very rare for a woman under 50, unless she is hypertensive or diabetic, to have coronary artery disease. It is said that actual myocardial infarction in women without these diseases below the age of 65 is quite rare.

On the other hand, on the basis of the frequent autopsy findings of coronary atherosclerosis in otherwise healthy young male Korean war casualties, Green in a recent paper in the A.M.A. states that all males over the age of twenty should be considered to be suffering from coronary artery disease.

Dr. Landmann has discussed the diagnostic symptoms of angina pectoris and the electrocardiographic confirmation where possible. Other causes of chest pain should be eliminated however. In speaking of angina pectoris, not anginal pain, it is understood that the patient is suffering from intermittent chest pain with complete relief of pain between attacks.

This fact alone will help eliminate patients who are acutely ill and suffering from such diseases as acute pericarditis, aortic aneurysm, spontaneous pneumothorax, mediastinal emphysema, epidemic pleurodynia and pulmonary emboli.

Especially to be considered are the recurring chest pains commonly seen in clinical practice. The first of these, and I think one of the most common, is that pain that is caused by skeletal, muscular, or nervous disorders. Included here are those of spinal root compression in the thoracic or cervical spine, either as a result of abnormal posture, osteoarthritis, scoliosis, or cervical disc. These all may cause irritation of a nerve root. This is not a diagnosis that can only be suspected but can often be confirmed by the finding of a tender area over the involved segment in the spine.

In addition, pain may be due to "trigger areas" of the various syndromes which go by the name of Tietze's Syndrome, the Xyphoid Process Syndrome, the Manubrial-Sternal Syndrome, the Costochondral Syndrome or the Parascapular Syndrome. In these cases there may be persistent or recurring chest pain and pressure on these areas will usually produce pain.

In addition, relief of these pains may be obtained very simply either by injection of local anesthetics or by ethyl chloride spray. Preeruptive herpes zoster should not be forgotten as a cause of pain and a postherpetitic pain may occasionally give diffculty in the differential diagnosis.

**Pain Is Persistent**

In considering these diseases or syndromes it should be noted that as a rule the pain is more persistent, it is slow to be relieved, and it is apt to be related to change of position but not to exertion in general.

As a rule relief may be obtained in these patients by correction of the underlying problem through corrective exercises, cervical traction, and other local means.

Another cause of chest pain is a thoracic tumor either in the lung itself or in the mediastinum. This pain has different characteristics and is apt to be quite persistent and lacking in relationship to exertion. This is the reason why many of us routinely x-ray the chest in patient's complaining of chest pain.

When minor abnormalities are noted in the chest x-ray, it is essential that follow-up films be taken.

A third cause of chest pain is pain referred from below the diaphgram from the gastro-intestinal and biliary tracts. Cardio-spasm or esophageal spasm will frequently cause pain over the lower sternum which may be very sudden in onset and which may stimulate anginal pain.

In these cases the pain is usually aggravated by eating or swallowing, may be temporarily relieved by belching, and is usually susceptible to diagnosis by esophagrams or other x-ray studies.

Hiatus hernia or diaphragmatic hernia is another disease to be considered. Here the pain is usually different, is usually aggravated by eating, may occur in the recumbent position, and may be relieved completely by the patient being upright.

Pylorospasm, peptic ulcer, and chronic gastritis are other causes of pain that may be referred into the lower chest and stimulate cardiac pain.

### Cholelithiasis & Cholecystitis

Cholelithiasis and cholecystitis should be considered. In this connection it should be remembered that Wakefield in 1940 showed that distention of the gall bladder at the time of cholecystectomy may produce inverted T waves which subsequently return to normal.

A careful history should elicit the expected symptoms and a cholecystogram may prove the diagnosis. It should be remembered that chronic cholelithiasis and cholecystitis may aggravate a pre-existing angina pectoris.

A fourth cause of chest pain is the psychogenic cause. This is difficult to arrive at because there are so many other possible causes. As a rule, however, the patient has other manifestations of anxiety and there may be unusual fear in the patient concerning the existence of heart disease. The latter may have been inadvertantly augmented by a previous examination by a physician or through a relationship to someone with heart disease.

It should always be remembered that the diagnosis of angina pectoris must rest on firm ground. David Davis in his monograph on the differential diagnosis of chest pain has said it is wise to beware of the diagnosis of atypical angina pectoris, because it usually indicates doubt on the part of the physician. It is also wise to remember that two diseases or two processes may exist in the same patient, as shown in the two cases presented tonight. One was a patient with a hiatus hernia, capable of producing chest pain, who subsequently developed a myocardial infarction. The second patient who undoubtedly had skeletomuscular causes of chest pain over a period of years, did develop angina pectoris and died presumably of a myocardial infarction.

**Dr. Ellis:**

Thank you, Dr. Angle. I was surprised in reading about this to find that there are some important things that a physician can do for a patient with anginal pain. There are not only conservative measures but in some instances surgical therapy may be of benefit. Dr. Richard Streeper is going to talk to us on this subject and also I've asked Dr. Streeper to discuss prognosis in persons suffering from angina pectoris. Dr. Streeper.

**Treatment and Prognosis: Dr. Richard Streeper**

### Medical Methods of Treatment

Obesity is one of the first factors to be corrected when present in a patient with anginal pain. If there is an excess intake of nicotine or alcohol, this should be corrected as much as possible.

Congestive failure is sometimes associated with an increase in the frequency of anginal attacks, hence proper management of congestive failure should be employed.

Conversely, at times in angina, congestive failure is associated with a reduction in frequency of anginal attacks. Any hypertension which exists should be treated. If there is anemia the cause should be established and treatment instituted. Thyroid disease can increase the frequency of attacks. This should be corrected. There are a number of other factors. The point is that if there is anything else contributing to the angina the first approach must be to try to correct these before attempting to control the symptoms specifically with drugs.

In so far as drugs are concerned, digitalis and quinidine are ineffective. Khellin was in vogue for a while, but most people are convinced that it is not effective in doses which can be tolerated. Most people are agreed that peritrate is not very useful. Nitroglycerine is still the drug of choice. This gives prompt relief which tends to last half an hour and there is very little danger in its use.

When treatment with nitroglycerine is insti-

tuted, the patient is cautioned to take the dose in a sitting position instead of in the erect position in order to avoid postural hypotension.

Aminophylline, and the other theophylline derivatives are not usually effective in angina pectoris. There has been some experimental work which would indicate that these drugs do improve coronary blood flow.

Heparin has been used by several groups, by injection twice weekly, and some reduction in frequency of anginal attacks has been reported. This work cannot be confirmed by all observers. It is an expensive form of therapy and certainly no more effective than nitroglycerine.

### Surgical Methods of Treatment

Beck proposed surgical procedures for this condition before general interest was aroused. His Beck I procedure is still in use. The Beck II procedure has been abandoned. The Beck I procedure involves the use of a rotary burr, scarifying the epicardium, partial ligation of the coronary sinus, and sprinkling of silica dust over the surface of the heart.

This has been modified by others. Some claims have been made for these procedures, but as in all of the surgical approaches that will be mentioned, one must consider the profound psychological effect of the operation in evaluating results and also one must consider the risk of the operation. The risk is still great and most of these operations have been reserved for the patient with angina decubitus or with very frequent attacks of angina.

Another surgical approach has been done by Weinberg which consists of implanting the internal mammary artery into a tunnel made by blunt dissection into the myocardium. This results in a large hematoma. Theoretically, there should be many collateral channels developed from this but autopsy specimens have not indicated the development of sizable channels. There have been a number of smaller channels developed but most of them would seem incapable of carrying significant blood flow. The initial favorable reaction noted in dogs in this procedure has been attributed by some to a reactive hyperemia which does not persist. Another surgical approach has been that of internal mammary artery ligation. This was practiced first by Lillehei. This was thought at first to be a startling discovery. Theoretically, this should increase the blood flow to the myocardium through the cardiophrenic artery. Coronary flow studies in dogs have indicated a minor increase. In evaluating the procedure simple skin incisions only have been made and these patients sometimes experienced marked relief of pain.

It is interesting in this respect that Glover ligated the right internal mammary artery and not the left, and relieved the pain on the right side, which indicates to me that there may be some rationale for the method aside from the psychological effect.

### Coronary Endarterectomy

More recently, Bailey's group in particular have performed coronary endarterectomy. There are others doing the same procedure. The mortality rate is still high for this operation. The idea behind this is that early in coronary disease there is segmental involvement of the coronary vessels. If there is segmental involvement, an endarterectomy is more apt to be effective.

Radiologic methods demonstrating the coronary circulation have improved remarkably in recent years, particularly with the use of timing devices so that the material can be injected during diastole. Beautiful visualization of the coronary vessels has been obtained. Unfortunately if a proximal part of a coronary vessel is occluded, one does not know whether the occlusion involves the entire length of the vessel or if there is segmental involvement.

Possibly with the passage of time and more case studies, such cases can be more properly selected for endarterectomy. At the present time, in my opinion, the surgical approach to coronary heart disease is still in an experimental stage.

### Treatment of Choice

At present I believe a treatment of choice for relief of intractable angina when conservative measures fail is the use of I 131 therapy. This is a safe procedure and is often effective. There are some theoretical hazards in the fact that serum cholesterol tends to rise in a hypothyroid patient, but the advantages of this procedure certainly outweigh the disadvantages.

I should mention in passing, posterior rhizotomy as a historical landmark. So many patients developed a radiculitis following this procedure that it

has been discarded. Sympathectomy has been of some benefit but the operative risk is high.

### Prognosis of Angina Pectoris

It is extremely difficult to say to any one coronary patient or anginal patient that he has so many years to live. There is a great weight of statistical evidence that can be utilized to more or less categorize these people into groups.

It is known that people who have strong family histories of coronary disease are apt to die at an earlier age than those who do not have a family history. The presence of other lesions such as syphilitic aortitis with angina is particularly ominous.

Rheumatic heart disease with aortic insufficiency and angina is also rather ominous. Calcareous aortic stenosis developing in an elderly patient associated with angina pectoris is associated with a poor prognosis.

People in their early 40's or less who have a coronary episode are much less apt to survive that attack than are people who are 65 or older. Here a mortality rate of three to five per cent in older patients may be compared with 35 per cent in younger patients.

Women tend to have a higher mortality than men.

### Study of 6882 Cases

In a followup study of 6882 cases of angina pectoris lasting from five to twenty-three years, about 15 per cent died in the first year after onset.

More and more evidence has accumulated to indicate the influence of prolonged anticoagulant therapy on prognosis in patients with angina pectoris.

The Suzman group followed 82 cases treated continually over a period of three months to six years, and compared their results with 88 untreated controls over the same period. They found the mortality was reduced from 33 per cent to 7.3 per cent. The frequency of recurrent coronary thrombosis was reduced from 24 to 17 per cent.

In another series Owren reported 128 cases of uncomplicated angina pectoris treated from one to five years with dicumerol or dindevan. There were only 10 instances of coronary thrombosis and the mortality during the first year of treatment was five per cent which represents a considerable reduction from the expected 15 per cent.

**Dr. Ellis:**

Dr. Bergere Kenny will summarize the discussion. From the way everyone has covered their subject this evening I wonder if there is anything left for him to say?

**Dr. Kenney:**

Seminar Summary: Dr. Bergere A. Kenney:

Actually there is still a lot to say. Driving up to Los Alamos the other day, trying to get a little peace and quiet and do some reading on this, I got stuck behind a truck going up a hill. This was a big van-like truck. I couldn't pass it because of traffic and then it pulled over to the side of the road, the truck driver got out and beat on the back of the truck with a long steel pole. Then he got back in, drove on for another mile, got out, and repeated this procedure. Finally he arrived at the top of the hill and again got out and beat on the back of his truck. I could have passed him at this point but I was so curious that I stopped and asked him what he was doing. He said "Well look, I'm driving a three ton truck and it is loaded with five tons of canaries. I've got to keep two tons of those birds flying all the time."

In a similar way, we have at least two tons of material on coronary disease and angina pectoris in the flying stage and this includes Beck's operations, endarterectomy and a variety of other things.

I think that one thing we tend to overlook in a discussion which is limited to the symptoms of a syndrome, as we have done tonight, is the nature of the underlying disease, which is afterall, coronary artery disease.

There have been a number of studies done all bearing out approximately the same figures, namely, that in autopsy material some 75 per cent of patients who had clinical angina pectoris also had two or three coronary arteries completely occluded. Many of these patients had normal electrocardiograms at rest. Occlusion of a coronary artery therefore does not necessarily imply the presence of a myocardial infarction. These arteries can occlude gradually and without clinical damage to the myocardium.

In one large series, the average number of occluded coronary arteries per heart in patients with clinical angina was 2.5. This certainly indicates that you can close off a coronary artery

without having an associated myocardial infarct. Furthermore, a myocardial infarct may develop without occlusion of a coronary artery. I'll mention this later.

Another thing, I think we tend to forget, is that the contraction of the heart muscle forcibly shuts off coronary blood flow during systole. Therefore, anything which affects cardiac rate (tachycardia, arrhythmia, etc.) will cut down on coronary perfusion because of the shortened diastolic time. Likewise, anything that will reduce diastolic pressure will tend to reduce coronary perfusion. Aortic insufficiency, for example, in which the mean diastolic pressure is low, is frequently associated with anginal pain. The same thing is true of shock and anything else that will lower the diastolic mean pressure.

Another factor to be considered, and this is more of a theoretical factor, is the matter of the diameter of a coronary vessel. The mean flow through a tube is dependent on several things, among which are the pressure differential between the ends of the tube, the length of the tube, and the viscosity of the fluid. The most important factor is the diameter of the tube, the flow being proportional to the radius multiplied to the 4th power. So if there is a slight decrease in diameter there will be a relatively marked decrease in the possible flow through the tube.

### Differential Diagnosis

In considering the symptoms and differential diagnosis of anginal pain, one is confronted not only with the differential points mentioned by Dr. Angle but also with the problem of variation of symptoms in true angina.

Work has recently been done in the course of open heart surgery in which electrocardiographic tracings were taken directly from the epicardium, using small cotton tipped electrodes.

In the anginal patient, the typical anginal patient with a normal electrocardiogram, there were abnormalities found in areas immediately involved, that is in areas of myocardial ischemia. These consisted of S-T depressions, the typical anginal pattern. And incidently in this group there were a number of areas of involvement, not just a single area.

It was also found that there was what the authors called a "variant form" of angina in which involvement was much more localized. It seemed to involve a single artery or branch of a single artery and over these areas there was S-T elevation, not depression. There was reciprocal S-T depression in the surrounding areas.

In these individuals the clinical course was characterized by a prolonged type of pain without relation to exertion, emotion, or the usual precipitating factors. When these patients developed coronary occlusion there resulted a single clinical infarct, so that in the so-called "variant form" you have a syndrome of an impending single occlusion, whereas in typical angina there is more of a diffuse process involving many areas of myocardial ischemia, without S-T elevation and the so-called "injury" pattern in the electrocardiogram.

### Stellate Ganglion Block

One thing I would like to ask about is stellate ganglion block or stellate ganglionectomy. These procedures are said to be effective in the relief of pain and their use is criticized on the basis that they take away warning signals from the individual. If you have a patient, with angina decubitus, it would seem to me that stellate ganglionectomy or stellate ganglion block might have a place.

In concluding my remarks I would like to mention a book I read recently in Los Alamos. It was written by Hans Selye, and the title was "The Chemical Protection of Cardiac Necroses." The book was very provocative, and I use the term provocative in the usual sense, meaning that I really didn't understand what he was talking about. His thesis appeared to be this: There are a number of conditions in which myocardial necroses occur. Diphtheria, digitalis intoxication, potassium deficiency, magnesium deficiency, and other agents may produce myocardial necroses. Characteristically they develop areas of local myocardial necrosis, which are followed later by replacement with scar tissue.

Selye has been able to produce this type of lesion by using several of the corticosteroids, in combination with certain salts of sodium, mostly the phosphates, the sulfates, or chlorides.

He was able to prevent this type of necrosis by using, in addition to the corticosteroids and the sodium salts, either potassium chloride or magnesium chloride. These latter salts appear to protect the myocardium from this type of necrosis.

Selye felt that in many myocardial infarctions—even fatal ones, that up to 30 per cent of these will not be associated with a clinical or anatomically demonstrable occlusion of a coronary vessel. He feels that there is something in the heart muscle in conditions of ischemia or anoxia, something of a biochemical nature, which results in exactly the same type of necrosis that one can induce by using a combination of corticosteroids and salts in animals.

In speculating as to the future, Selye felt that there may be a way of finding the physiologically balanced electrolyte pattern by which a certain amount of this damage can be avoided. So perhaps the future of coronary artery disease and angina pectoris will come to the door of physiologists, the biochemists, and the internists rather than the surgeon.

**Dr. Ellis.**

We have a few minutes left for discussion. Dr. Derbyshire, can you answer Dr. Kenney's question regarding stellate ganglionectomy for anginal pain?

**Dr. R. C. Derbyshire:**

All I can say about that is that I wish the problem of intractable angina could be solved by such a relatively simple procedure. Unfortunately, it is not this simple as stellate ganglionectomy alone will not control the pain.

As you know, Smithwick has written an excellent book on the sympathetic nervous system and I think it is still valid. He was doing a much more extensive operation than stellate ganglionectomy. He not only took out the stellate ganglion, but the entire upper chain, including the first, second, third and fourth thoracic ganglia.

I do not believe that stellate ganglionectomy is an adequate operation for anginal pain. In the case of patients who have unbearable pain, and are completely incapacitated by this pain, the more complete operation seems justifiable. My understanding is that even with this operation only about 70 per cent of these patients are relieved of their pain.

I would like to ask three questions. #1—What is the rationale of heparin therapy for anginal pain? #2—How is it given? #3—Dr. Streeper, would you say a little more about coronary thromboendarterectomy. I am interested in this because of Longmire's work. If I recall this correctly, in his original report he had 4 or 5 cases, one of whom died postoperatively and in the remainder he thought his results were promising.

**Dr. Streeper:**

In answer to your question about heparin, it's given intramuscularly once or twice weekly in dosage of 50 to 100 mgs. According to Graham there has been a marked reduction in the number of attacks of angina pectoris in 55 of 59 patients. As mentioned, these results have not been uniformly confirmed.

Longmire, in Los Angeles, and Baily, in Philadelphia, have both been doing coronary endarterectomies. Thus far, their series is small and it is too early to be sure of the surgical risk and too early to know the ultimate benefits.

**Dr. Richard Angle:**

Dr. Longmire has recently reported an extension of his series of thromboendarterectomy patients and he reports a continuing mortality of around 35 per cent. An interesting new development here is the use of a special polyethylene catheter with an overlapping loop in the end which approximates the circumference of the root of the aorta. Threaded through the femoral artery, this delivers diodrast to the orifices of the coronary arteries in high enough concentration to produce excellent visualization of the arteries.

Another aspect of the treatment of angina pectoris is the anticipatory use of nitroglycerine. When an act is to be undertaken which usually precipitates angina, nitroglycerine is taken beforehand in order to prevent this attack.

One last comment is that which concerns a recent paper in the J.A.M.A. which gives a very preliminary report on the use of antimalarials such as plaquenil as a desludging agent in certain thrombotic disorders. Good results in angina were reported but the test period was too brief to be significant.

**Dr. Ellis:**

If there are no other questions or comments, this will conclude our seminar this evening. Our next seminar will be about Hemolytic Disease of the Newborn.

ORIGINAL ARTICLE

# Peptic Ulcer and Allied Diseases

## *Treatment with a Single Preparation*

ARNOLD ALBERT, M.D., *San Antonio*

The diverse etiological factors and the multiplicity of symptoms involved in peptic ulcer dictate for practical therapeutics the use of a combination of drugs for treatment, although ideally all medication should be used individually for its specific action. Most patients with peptic ulcer have a history of recurrent gastric disturbances of several years' standing. During the periods of attack, such symptoms as dyspepsia, belching, flatulence, heartburn, constipation, insomnia, irritability, nausea, vomiting, pain and loss of weight commonly occur.

Among the well known etiological factors which play a role in this complex disease are erosion, both mechanical and chemical; local tissue resistance; systemic and constitutional diathesis; neurogenic and personality factors. Modern peptic ulcer therapy is, therefore, directed in three channels:

**1)** The neutralization of gastric acid secretions with antacids.

**2)** The reduction of vagotonia with anticholinergics.

**3)** The control of emotional stress and stomach activity by mild sedation.

The purpose of this paper is to report the excellent clinical results obtained with a single preparation combining aluminum hydroxide gel, ambutonium bromide, butabarbital and a constipation-inhibiting dose of magnesium hydroxide. In the neutralization of acids, aluminum hydroxide gel has been widely used because it has a tendency to coat the devitalized and ulcerated areas, thus providing physical protection against further digestive trauma.

At the same time it buffers the stomach pH between three and five, inactivating pepsin and relieving pain. The atropine-like action of ambutonium bromide has been reported.[1,2,3] This anticholinergic action reduces gastric juice volume and acidity. The concentration of butabarbital in the preparation was selected as sufficient to allay anxiety and deal with insomnia without causing daytime drowsiness.

### Materials and Methods

The treatment to be described was used in a group of 28 patients, 16 males and 12 females, ages 32 to 74 years. Thirteen of these had duodenal ulcer, confirmed by x-ray; three had gastric ulcer, one recurrent and two confirmed by x-ray; four had gastritis without evidence of ulcer on x-ray; and eight suffered from various functional diseases (4, pylorospasm with hyperacidity syndrome; 2, spastic colon; 1, diverticuli of the descending portion of the duodenum; 1, diaphragmatic hernia.)

The medication* was an aluminum hydroxide suspension containing in each teaspoonful 2.5 mg. ambutonium bromide, 8.0 mg. butabarbital, and the equivalent of one-quarter teaspoonful of milk of magnesia. For the majority of the patients the prescribed dose was one teaspoonful taken four times a day, before meals and at bedtime. In one case the dose was increased to one and one half teaspoonfuls, and in three cases to two teaspoonfuls.

*Aludrox® SA-Wyeth Laboratories, Philadelphia, Pennsylvania

A bland diet was recommended for patients with ulcers, gastritis and hyperacidity; the diets of other patients were restricted according to their particular needs. Increased rest was suggested and the patients were advised in resolving problems which caused anxiety. The patients were examined after one week, four weeks and eight weeks and thereafter at varying intervals.

## Results

Twenty-seven of the 28 patients included in the study tolerated the medication well. One patient discontinued its use because of minor side effects (see below). The clinical response in the group is summarized in Table I.

TABLE I

Clinical Response Observed in Twenty-seven Patients

| Diagnosis | Number of Cases | First Week | | | | Fourth Week | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Excellent | Good | Some | None | Excellent | Good | Some | None |
| Duodenal Ulcer | 12 | 5 | 4 | 2 | 1 | 11 | | | 1 |
| Gastric Ulcer | 3 | 1 | 2 | | | 2 | 1 | | |
| Gastritis | 4 | 3 | 1 | | | 3 | 1 | | |
| Functional Diseases | 8* | 6 | 1 | | | 6 | 2 | | |
| Totals | 27 | 15 | 8 | 2 | 1 | 22 | 4 | | 1 |
| | | (57.7%) | (30.8%) | (7.7%) | (3.8%) | (81.5%) | (14.8%) | | (3.8%) |

* One patient in this group did not report for the first week check-up.

Twenty-three patients (88.5 per cent) reported good or excellent response after the first week of therapy. After four weeks, 26 patients (96.3 per cent) considered their response good or excellent. One patient reported no improvement after one week and was still unimproved after four and eight weeks of therapy. The treatment was discontinued at this time. This patient had a duodenal ulcer and diverticulosis of the colon.

Three patients with a diagnosis of duodenal ulcer were x-rayed after eight weeks of therapy. In one patient the ulcer was completely healed and in two there was partial healing. Three additional patients from this group were x-rayed after twelve weeks of therapy. One patient was examined during surgery for removal of common duct stones at this time. In all four cases the ulcer had completely healed. Two other patients in the group were completely asymptomatic; clinically they appeared to be healed.

The response of one patient with gastric ulcer deserves special mention. When first seen, this patient had lost forty pounds and had hypoproteinemia from vomiting and inability to retain food. X-ray revealed a marked ulcer crater in the lesser curvature (Fig. 1). Medical therapy was instituted preparatory to surgery.

*Figure 1. E. B., X-ray showing gastric ulcer before treatment.*

At the end of one week he reported his clinical response as good and after four weeks as excellent. The patient regained his weight. There was no bleeding after the first week; his blood count returned to normal after oral medication with iron, without transfusion. Eight weeks after therapy began, x-ray showed tremendous healing (Fig 2). Plans for surgery were abandoned and the patient was continued on maintenance therapy.

The suspension was considered "excellent" with regard to taste, color etc., by 23 of the 28 patients; three others considered it "good;" one considered it "fair;" and one considered it "poor."

*Figure 2. E. B. X-ray showing marked healing after eight weeks of therapy.*

Twenty-four of the patients continued the medication as maintenance therapy.

Minor side effects were reported by only two of the 28 patients studied. One suffered from nausea and faintness during the first three days of therapy. This patient tolerated the aluminum hydroxide gel with magnesium hydroxide, without the ambutonium bromide and butabarbital. The other patient suffered from constipation. However, he accepted the combined medication as "excellent" and has continued its use as maintenance therapy; cascara sagrada was prescribed to relieve his constipation.

Two patients with duodenal ulcer attempted to stop the medication during maintenance therapy. One reported a recurrence of indigestion and the other a recurrence of hyperacidity. In both cases the symptoms were relieved when medication was begun again. Two patients with pylorospasm elected to interrupt therapy and both complained of a recurrence of symptoms during the period of interruption.

## Comments

The excellent clinical response observed indicates that the combination of aluminum hydroxide, ambutonium bromide, magnesium hydroxide, and butabarbital, in the combinations used, produced an effective, single preparation for the treatment of peptic ulcer and other upper gastrointestinal diseases. Prompt relief from symptoms was experienced by the majority of the patients. This was probably reflected in their acceptance of the medication as a palatable preparation. The simplicity and convenience of this form of therapy appeals to both the doctor and the patient.

Both the antacid component and the anticholinergic drug contribute to the singular effectiveness of this combination in controlling gastric hyperacidity. Ambutonium bromide exerts an atropine-like action on the effector organs innervated by the parasympathetic system and a blocking effect on the autonomic ganglia of the sympathetic and parasympathetic systems.

These two effects are anticholinergic and both combat the vagotonia which is characteristic of the ulcer diathesis. The influence of vagotonia on both acidity and gastric activity is well recognized.

## Summary and Conclusions

(1) The effectiveness of a combination of aluminum hydroxide, ambutonium bromide, magnesium hydroxide, and butabarbital in the treatment of peptic ulcer and allied diseases was studied in twenty-seven patients. (2) The preparation relieved symptoms promptly in 26 of the 27 patients. (3) Minor side effects were reported in two of the 28 patients who began treatment. These included nausea, faintness and constipation. (4) The preparation was well accepted by the patient. The simplicity of this form of medication appealed to both the patient and the doctor.

2714 S. Presa St.

### References

1) Cheney, L. C. et al.: Basic Amides as Antispasmodic Agents, J. Org. Chem. *17*: 770-777, May, 1952.
2) Judge, R. D., Bolt, R. J., Hirschowitz, B. I. and Pollard, H. M.: Pharmacological Evaluation of BL700B, J. Lab. & Clin. Med. *47*: 950-959, June 1956.
3) Rosen, H., Blumenthal, A., Nead, M. H., Tislow, R. and Seifter, J.: Gastric Juice Studies in Rats, Federation Proc. *16*: 331, March, 1957.

# Female Genital Tuberculosis

JAMES RODERICK MORGAN, M.D., *El Paso*

Pelvic infections by acid-fast organisms occur much more frequently than is generally appreciated. (Chart #1) Many cases are discovered at infertility clinics where routine endometrial biopsies are done.

**Clinical Features and Symptoms:**

The organs involved in genital tuberculosis are:

Fallopian Tubes; 90 per cent

Uterus (endometritis) — (secondary spread from tube); 50 per cent

Ovaries; 25 per cent

Cervix, Vagina and Vulva; Rare.

Symptoms include infertility, abdominal pain, and heavy or irregular bleeding.

A. **Infertility:** Although a few pregnancies have occurred in patients with genital tuberculosis, sterility is the rule and no successful pregnancy has been recorded after a definite diagnosis of genital tuberculosis has been made. Ectopic pregnancy and abortions are common with pelvic tuberculosis.

B. **Abdominal Pain:** While this usually takes the form of chronic lower abdominal discomfort, this symptom is not prominent in early cases; therefore, those patients who have this symptom may be regarded as having a more advanced disease. Severe and acute pain is usually associated with pyrexia and with a mixed infection of coliform and acid-fast organisms. Such acute attacks commonly follow uterine curettage or a tubal patency test.

C. **Menstrual Aberrations:** The menstrual periods are normal in about one-half the patients; in about 40 per cent of the cases there is menometrorrhagia; 10 per cent manifest oligomenorrhea or amenorrhea. Dysmenorrhea is not a feature of the disease.

Poor general condition is an infrequent finding; undue fatigue and lassitude, loss of weight and appetite, and low-grade fever are noted in only about five per cent of the cases.

**Chart No. 1**
**Incidence of Female Genital Tuberculosis**

| | No. of Patients | No. of Genital TBs |
|---|---|---|
| **Jedbury (1950)** | 1,168 | 2.1 |
| **O'Brien & Lawlen (1947)** | 898 | 1.9 |
| **Halbrecht (1946)** | 820 | 5.5 |
| **Sharmon (1952)** | 3,255 | 5.3 |
| **Hayne: (1952)** | 200 | 4.0 |

**Diagnosis of Genital Tuberculosis.**

There are no clinical features that will establish the diagnosis and frequently no physical signs to aid the physician. The pelvis may appear perfectly normal on physical examination. In some of the more advanced cases, hard masses, which are not tender, may be felt behind the uterus. Peritoneal irritation is shown by tenderness, resistance to palpation, "doughy" abdomen, or ascites.

There are, however, certain clinical clues which are helpful in diagnosis, as follows:

1. Family history of tuberculosis.
2. Past medical history of tuberculosis.
3. Presence of tuberculosis outside the pelvis.
4. History of pelvic disease.

**Laboratory aids: 1. X-ray.** Since pelvic tuberculosis is almost always secondary to disease elsewhere, and since the lung is the commonest primary site, the patient should be examined radiologically for evidence of a pulmonary lesion.

In 1945, Magnusson described the radiographic appearance of tuberculous salpingitis as seen in X-ray: "The isthmus of the tube is rigid with a smooth outline and gives the picture of a lead

pipe, terminating in the form of a dilated bulb where it joins the ampulla. Calcification in the tube is sometimes seen. Coexistent endometritis can be diagnosed, if it is far advanced, by the irregular saw-toothed outline of the uterine cavity."

2. **Histological and Bacteriological:** Two methods of bacteriological examination are available: culture and animal inoculation.

Staining the smears of the endometrium or of vaginal discharge gives poor results because of the few bacilli that are usually present. Cultures of the endometrium, vaginal secretion, or menstrual blood can be made on suitable media (Dorset egg or Lowenstein-Jenson). Because fresh tubercles are formed during the growth of the endometrium at each cycle, the endometrial biopsy should be made in the week preceding the onset of the menstrual period. Cultures are examined weekly up to two months. For animal inoculations a small piece of endometrium is placed in the thigh muscle of a guinea pig through a small incision. The animal is killed six weeks later and smears are taken from the regional and lumbar lymph nodes and the spleen.

Whether isolated by culture or by animal inoculation, the organism is typed as to human or bovine strain, and sensitivity to antibiotic drugs is determined.

## Treatment

As most, if not all, patients have active pulmonary disease, treatment for such disease should take precedence over treatment for the pelvic disease. Methods of treatment include:

1. Sanatorium Treatment
2. Chemotherapy
3. Surgery
   a. Conservative
   b. Radical
4. Irradiation

1. **Sanatorium Treatment:** Adequate mental and physical rest, together with proper nourishment, improves the general health of the patient. Few cases of pelvic tuberculosis are cured with this treatment alone.

2. **Chemotherapy:** Whether drug and sanatorium treatment will finally arrest the disease has not been established. It has been shown, however, that this treatment makes patients safer for conservative surgery rendering radical surgery less necessary. Investigations and follow-up of cases are being done now and early reports indicate that chemotherapy will give long term cures without surgery.

**Chart No. 2**

**Results of Chemotherapy in Tuberculosis Endometritis up to Three Years After Treatment. (Millar—1954), (Streptomycin and PAS)**

| Length of time after treatment | No. of cases | Endometrium Positive | Endometrium Negative | Per cent Negative |
|---|---|---|---|---|
| 1 year | 36 | 5 | 31 | 86 |
| 2 years | 32 | 3 | 29 | 90.6 |
| 3 years | 19 | 2 | 17 | 89.5 |

There is no standard formula for chemotherapy, but usually the patient is given 1 gm. daily of streptomycin (calcium chloride complex IM) for 84 days and 3 gm. para-aminosalicylic acid (PAS) (sodium salt) q.i.d. orally for 84 days.

Similar results are reported using various combinations of streptomycin, PAS (para-aminosalicylic acid), and INAH (Isonicotinic acid hydrazide), but the best combination of these have yet to be worked out.

3. **Surgical Treatment:** Prior to chemotherapy, genital tuberculosis was either left untreated or radical surgery was done; incomplete removal of the affected organs was (and still is) likely to result in the development of fistula (usually fecal fistulas).

Indications for Hysterectomy and Bilateral Salpingo-oophorectomy:

(1) Postmenopausal patient with troublesome symptoms.
(2) If adnexal masses appear to be increased in size during conservative treatment.
(3) If there are fistula which fail to heal (incision of tract and resection of affected section of bowel).
(4) Endometrium which is positive after medical treatment.

Hysterectomy in the younger female is more liberally indicated as successful pregnancy associated with this disease is extremely rare.

4. **Irradiation:** Pelvic X-ray treatment in small doses used to be given, but this form of treatment has been abandoned.

Bibliography: Furnished by author on request.

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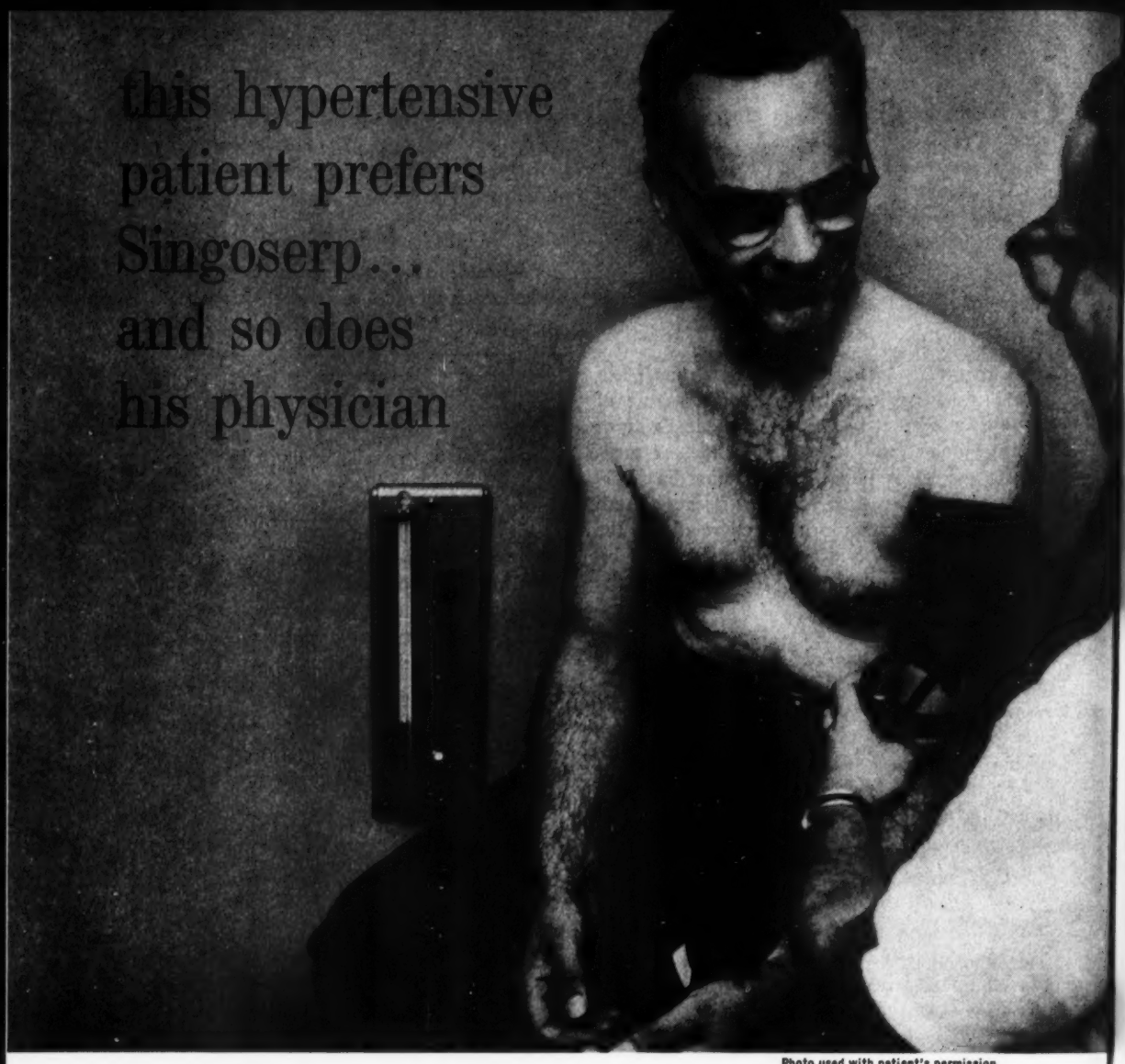
# this hypertensive patient prefers Singoserp... and so does his physician



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# Clinical Pathological Conference

R. E. Thomason General Hospital, El Paso

Case No. 1472, August 18, 1960

F. P. Bornstein, M.D., *Editor*

Presentation of case by C. M. Stanfill, M.D.

**History: Dr. Nathan Kleban:**

Confusion and fever provoked admission of a 59-year-old married Anglo-American woman to the hospital June 24, 1960.

Obstruction to nose breathing was not relieved by removal of a polyp in May, 1954. A granular, slightly friable mass in the posterior nasal space was visible through both nostrils when the patient was re-admitted to a hospital in December, 1955. There was also a small polyp in the region of the left middle turbinate. Pathological diagnosis of tissue removed was nasal polyp and epidermoid carcinoma. Decision to treat the tumor by excision only was reached after pathological consultation with Walter Reed Army Hospital and opinion that the growth was not of an invasive type.

Tumor tissue was again removed in June, 1956. Pathological diagnoses were intraepithelial carcinoma; squamous cell carcinoma, invasive, bone tissues with invasive tumor.

Psychotic illnesses were treated with electroconvulsive therapy in October, 1956, August and December, 1957, and May, 1958.

## Pathological Diagnosis

Pathological diagnosis of tissue removed in February, 1958, was epidermoid carcinoma associated with carcinoma in situ. In July, 1958, tissue removed from the region of the sphenoid sinus was called papillary carcinoma.

Seropurulent nasal drainage was seen in March, 1959, when the patient was treated for an agitated depressive reaction following the death of her dog. Sinus and skull x-ray films were negative.

The patient went to the M. D. Anderson Hospital and Tumor Institute of the University of Texas at Houston on September 8, 1959, for evaluation and treatment after receiving Cobalt therapy at some time subsequent to her last surgery. Nasal polyps were seen to fill the left nasal cavity. Surgery done on October 16, 1959, consisted of partial resection of left maxilla with curettement of maxillary, sphenoid and ethmoid sinuses, plus cauterization, Papillary-like tumor tissue filled the left maxillary antrum. There seemed to be extension of tumor tissue into the left sphenoid sinuses. Pathological diagnosis was squamous cell carcinoma, invasive, arising in association with epithelial papilloma.

## Irradiation

From November 3 until December 9, 1959, the patient received irradiation with tumor dose of 5000 r to a single left lateral 8 cm Betatron port. Granulation tissue thought to represent residual tumor on the posterior pterygoid and pharyngeal wall areas extending to involve the surgical margin and soft and hard palate was seen at the beginning but not at the end of the treatment period. Radiation reaction was relatively mild. A pressure ulcer of the soft palate corresponded to the posterior margin of the prosthesis on the left.

On February 10, 1960, at the M. D. Anderson Head and Neck Clinic the patient was considered

free of disease, although crusting was present. She had not used the prescribed nasal irrigating kit or the oil pack at night.

Complaint of headache, given a diagnosis of post-operative neuralgia, hospitalized the patient in March, 1960. She failed to keep a return appointment at the Anderson Clinic on June 8.

The patient had had one pregnancy and had one child. Her uterus had been removed in 1924 at the age of 24.

Family history was not pertinent.

**Physical Examination:**

T. 102 (R); P. 70; R. 20; B.P. 130/84.

The patient was confused, disoriented, noisy, incontinent of urine, refused to eat, moaned. Odor of breath was fetid. A palate prosthesis was in place. She was unable or unwilling to stand. No other abnormal findings were described.

**Hospital Course:**

Tetracycline 100 mg. was given 3 times I.M. on the first day. For the next 3 days she was given 500 mg. of tetracycline orally t.i.d. Temperature was normal by the second day. The patient improved to some extent, began to eat, got out of bed, talked, but remained confused, voided in bed. She was given vitamins, protein supplement and a tranquilizer. On July 2 an anti-convulsant drug was started and the tranquilizer stopped. The nose was irrigated with salt water.

Temperature rose to 102 (R) on July 14. She became restless, apprehensive, hypotensive, vomited, complained of pain in the lumbar region. Tetracycline 100 mg. was given q.i.d. starting July 15. Stupor and tremors of the trunk were noted on July 16. Rigidity alternated with more severe tremors of neck and body. There were no lateralizing neurological findings.

Spinal paracentesis was done on July 19. Pressure was not recorded. Fluid was turbid. Parenteral chloramphenicol (Chloromycetin) was prescribed. Temperature climbed to 104 (R). Breathing became labored. Stupor changed to coma. The patient died July 23, 1960.

**Laboratory Findings:**

**Blood Counts: 6-24-60** — Hb. 12.4 gms., Ht. 44%, WBC, 10,800, Juveniles 1, Stabs. 6, Segs. 77, Lymphs. 15. **7-11-60** — Hb. 12.9 gms., Ht. 42%, WBC 12,300, Stabs. 11, Segs. 66, Lymphs. 23. **7-14-60** — Hb. 11.7 gms., Ht. 36%, WBC 19,200, RBC 3.95, Juveniles 1, Stabs. 13, Segs. 63, Lymphs. 23. **7-20-60** — Hb. 11.5 gms., RBC 3.68.

**Urinalyses: 6-27-60** — Amber, turbid, alkaline, S.G. 1.007, Albumin 1 +, Sugar negative, WBC 4-5, few round ep. cells, amorphous and triple phos. crystals. **7-14-60** — Amber, cloudy, acid, S.G. 1.020, Albumin 1 +, sugar negative, lt. amorph. sed., WBC 0-1, RBC 16-18, ep. cells 2-3, occ. granular casts, bacteria light (rods). **7-15-60** — Amber, turbid, alkaline, S.G. 1.006, Albumin 2+ sugar negative, WBC 1-2, RBC 0 — few squamous ep. cells, amorphous and triple phos. crystals.

**Chemistry: 7-5-60** — Chlorides (as Na Cl) — 108 mEq/L; Potassium—3-1 mEq/L; Sodium—142 mEq/L. **7-15-60** — Calcium—4.5 mEq/L (Normal 4.5-5.5 mEq/L).

**Serum magnesium: 7-6-60** — 1.3 mg.%.

**Blood Culture and stain for bacteria: 7-19-60** — Gram stain, no bacteria seen. Culture, no growth.

**Spinal fluid: 7-19-60** — WBC 277, Polys 263, Lymphs. 14, Sugar 50 mg.%. Total protein 245 mg.%, Globulin 4+, no bacteria seen on smear, no A.F.B. seen, many WBC seen, culture showed proteum rettgeri.

**Serology: 6-27-60** — Negative.

**Dr. Bornstein:**

If you read the protocol you will see that there was considerable discussion about this case with the Walter Reed Hospital Armed Institute of Pathology. In connection with this the whole problem of non-invasive carcinoma had to be considered. With this in view, I have reviewed all the slides we have on this patient. The first biopsy in 1954 shows a marked pseudo-epitheliomatous hyperplasia with some cellular irregularities. A year later the cellular anaplasia has become more outspoken but no real invasion was demonstrable during the life time of the patient.

**Clinical Discussion — Dr. C. M. Stanfill:**

These carcinomas of the nose and naso-pharynx are not uncommon. I imagine in this neighbor-

hood we will find about 20 to 30 every year. For people and this is really important, who examine noses, it is of importance to know what to do with the so-called polyp. I don't think anyone can look at a polyp and tell whether it is going to be benign or malignant. Anybody who has ever removed a great number of polyps and thinks he has removed a benign one, will occasionally be surprised by a report of malignancy.

Many people treat the so-called allergic polyps with cortisone and it would be good if everybody would have a careful look so that one does not treat a malignant case with cortisone. Now this lady apparently had a nasal polyp. It was biopsied and was benign. Three years later from the protocol she apparently had a biopsy from the nasopharynx which are reported as cancer in situ. It is a well known fact that many of these nasopharyngeal cancers can be less than the size of an eraser head and still they can produce bilateral neck nodes and spread all over.

### Potential Carcinoma

I think that this woman should have been treated vigorously even with a diagnosis of only potential carcinoma. The further course of this thing is apparently typical. She apparently died of an extension through the sphenoid ridge and either developed a fatal brain abscess or meningitis. Apparently she developed a naso-pharyngeal cancer which spread into her ethmoid sinuses, and antra.

The surgical and medical treatment of these naso-pharyngeal cancers, is almost totally hopeless. Maybe five to ten per cent are being salvaged, most of them with Cobalt and deep x-ray therapy. If you have a naso-pharyngeal cancer on the lateral wall, then you would be justified in doing a neck resection provided you were sure that you could eradicate the primary lesion, but most of them and this is a peculiar feature of the malignancies of the naso-pharynx that the lymphocytes will permit immediate bilateral spread.

I can't see that there would be much point in doing a bilateral neck dissection, because there ususally is spread into the ethmoids and into the sphenoid ridge. The malignant tumors of the nasopharynx have a proportion of 2 cases of carcinoma to 1 of sarcoma.

### Transitional Cell Type

The fairly common carcinoma is your transitional cell type which is the so-called Schmincke tumor. The Schmincke tumor, I guess, has become more famous through the fact that Babe Ruth had one. The tumor metastasized early to his neck nodes and to his brain.

There is another important point if anybody is going to make a diagnosis of nose and throat pathology, they should always examine the nasopharynx. This is done easily with naso-pharyngeal mirror or with a nasopharyngoscope. I think if people are going to check a patient from the nose and throat standpoint, they ought to be sure that the nasopharynx is free of tumor. It is hard to see up there sometimes, but with a little practice most anybody can look up there and after you have looked at several and seen what is normal, you can tell that something is abnormal if you just look.

**Dr. K. Zolfoghary:**

I remember I have seen two cases of nasopharyngeal tumor and both of them started from the nose and then spread backward into the eye and outward into the base of the brain. Unfortunately I have seen enucleation of the eye in the hope of completely curing the patient, but often some of the tumor was left behind and we had to go in and remove an extensive tumor in the temporal region. This tumor was squamous cell in type and the patient had extensive Cobalt therapy. I think she died of meningitis with extending infection from the nasopharynx upwards. In the other case they did neck dissection but some part of the tumor remained. Again Cobalt therapy was used but still the tumor extended to the base of the brain, actually extending through the meninges and into the tentorium. At autopsy the tentorium was completely invaded by the tumor, of course the bone also was involved.

**Dr. I. A. Budwig:**

I just want to mention a little-bit about the spinal fluid, and I was wondering if occasionally you can diagnose a tumor of the brain by spinal fluid, and it would be interesting if someone had looked at the slide, who was trained, at the smear on the spinal fluid, it is reported as 277 white blood cells. I am sure she had a secondary menin-

gitis due to invasion of the tumor, but it would have been interesting to see if there were any carcinoma cells in the spinal fluid. I have seen two children that were brain or spinal cord tumors, and were diagnosed from the spinal fluid.

**Dr. Bornstein:**

They probably had ependymomas.

**Dr. H. M. Gibson:**

I would like to raise the question of false positive biopsies. I raise this question because a friend of mine who lives in one of the neighboring towns was discharged from the armed forces as a hopeless, inoperable, far advanced carcinoma of the naso-pharynx. This was approximately in 1946. The patient is alive and completely well. The other is an in-law of mine that was also diagnosed in one of the institutions to the north of us and returned and was re-operated for an additional area and was told that it was out of control and yet as far as the nasopharynx is concerned, this is completely normal. Now we run into this problem of pseudopositive biopsies in the field of urology fairly often and I would like to ask the pathologist and Dr. Stanfill just what part of the possibility of a false positive biopsy might play in prognosticating some of these cases.

**Dr. Bornstein:**

Well, I can only speak for myself, I don't think that you have gotten a false positive from me yet?

**Dr. H. M. Gibson:**

No, I have not, Fred.

**Dr. Bornstein:**

I showed that first slide in the very beginning because it demonstrates the extremely difficult problem to evaluate these lesions. Certainly we all make mistakes, on the other hand there is a small group of really well proven histologically malignant tumors which have regressed spontaneously, you can't get away from it, they do exist. I would say that there is more soul searching involved in the nasopharyngeal tumors, of the squamous type. The so-called Schmincke tumors permit a clear cut diagnosis, but the borderline from pseudoepitheliomatous hyperplasia to active malignancy is an extremely difficult one, that is why I picked the cause and showed the pictures.

**Clinical Diagnosis:**

Tumor of nasopharynx.

**Dr. Stanfill's Diagnosis:**

Tumor of nasopharynx with intracranial metastases.

**Dr. Bornstein's Diagnosis:**

**A.** Carcinoma of the naso-pharynx with direct extension into the brain.

**B.** Secondary brain abscess and empyema of ventricles.

*Figure 1*

**Pathological Discussion:**

The autopsy of this patient showed an aspira-

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tion bronchopneumonia and extreme emaciation. There was a large prosthesis covering the maxilla, when this prosthesis was removed there was a tremendous hole and an ulcerated lesion with necrotic tissue around it. The main lesion was in the brain. The brain was covered with thick pus.

Figure 1 shows the brain in a rather unconventional manner. I so to speak, sort of took off the roof of the ventricles to show the centricles as seen looking down on them from above. Both ventricles were filled with yellowish-green pus. We did not find any evidence of tumor in the brain, but meningitis and emphyema of the ventricles. On sectioning the brain transversely, I also found an abscess in the frontal lobe. As far as the tumor is concerned, I took a section from the necrotic material at the edge of the prosthesis (Fig. 2). If you remember the previous

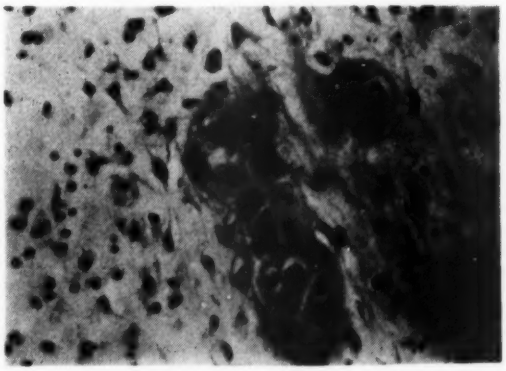

*Figure II*

slides, they showed a fairly well differentiated lesion, while on this slide one sees obviously malignant invasive primative clusters right at the edge of this granulation tissue.

### Meningest Invaded

In addition the tumor had invaded the meningest which seems to be a fairly common occurrence in nasopharyngeal tumors. There is a special tendency to invade the ganglia and peripheral myelineated nerve at the base of the brain. In summary then, this is a case of carcinoma of the pharynx which lasted for about 6 years from the time the case was first operated and showed a non-invasive carcinoma. In the course of time the carcinoma became invasive and in spite of extensive surgery and radiation, penetrated to the brain where it caused an infection and brain abscess.

## Announcement for General Practitioners and Internists

Applications for charter membership in the American Society of Diagnostic Radiology are now being received. Membership is open to general practitioners and internists who do or may desire to do some types of diagnostic radiology in their offices.

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## Is Anybody Listening?

All of us like to think that we operate around 90 per cent of our efficiency, but the cruel fact is that we are only 25 per cent efficient in a thing we do 45 per cent of the time. That thing? The matter of listening.

According to Carl I. Flath, F.A.C.H.A., John G. Steinle & Associates, New York, researchers have found that white collar workers, including management, spent 45 per cent of their working time listening, but that their listening efficiency is only 25 per cent of capacity.

The researchers also found in studying 100 companies that when directors sent a message down through levels of management and workers, vice-presidents understood 67 per cent of what was said, general supervisors 56 per cent, plant managers 40 per cent, foremen 30 per cent —and workers less than 20 per cent!

*—Hospital Digest*

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